IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF OHIO
WESTERN DIVISION

Carolyn Baker, et al.,	)	
	)	
Plaintiffs,	)	Case No. 1:05-CV-227
	)	
vs.	)	
	)	
Chevron USA, Inc., et al.,	)	
	)	
	)	
Defendants.	)	

O R D E R

This matter is before the Court on Defendant Chevron USA, Inc., et al.'s Motion for Summary Judgment (Doc. No. 197) and motion under Daubert to Exclude Expert Evidence from James Dahlgren, M.D. (Doc. No. 203), Plaintiffs' memorandum in opposition to Defendant's Daubert motion (Doc. No. 221), Defendants' Objections to Evidence Submitted by Plaintiffs in Opposition to Defendants' Motion for Summary Judgment and Motion to Exclude Expert Evidence from James Dahlgren and Nicholas Cheremisinoff (Doc. No. 236), Defendants' Reply Brief in Support of its Daubert Motion (Doc. No. 237), Plaintiffs' Response to Defendants' Objections (Doc. No. 240), Plaintiffs' Supplemental Response to Defendants' Objections (Doc. No. 246), Plaintiffs' Supplemental Brief Supporting Their Opposition to Defendants' Motion to Exclude the Opinions of James Dahlgren (Doc. No. 375), and Defendants' Response to Plaintiffs' Supplemental Brief (Doc. No. 376). For the reasons that follow, Defendants' motion to exclude the opinions of James Dahlgren is well-taken and is

GRANTED. As a consequence of granting that motion, Defendants' motion for summary judgment is also well-taken and is **GRANTED.**

I. Preliminary Background Information

As explained by the Court in earlier orders, Plaintiffs in this case are residents of the villages of Hooven, Ohio and Cleves, Ohio who assert claims for personal injury and property damage allegedly resulting from the Gulf Oil refinery, now owned by Defendant Chevron USA, Inc. ("Chevron").[1] For case management purposes, the matter was bifurcated between personal injury claimants and property damage claimants. The parties were permitted to select bellwether plaintiffs for each trial group. This aspect of the case concerns the claims of the bellwether personal injury claimants, Mary Etta Brown Greener, Michelle Schrader, Jean Runck, and Carol Lipscomb.

Hooven and Cleves are small towns located in the western part of Hamilton County, Ohio. Gulf Oil Company operated a gasoline refinery which was situated on the eastern edge of Hooven from 1930 to 1985. Gulf also refined diesel fuel, jet fuel, and fuel oil at the refinery and operated an asphalt plant at this location. Gulf and Chevron merged in 1985 and Chevron closed the refinery in 1986. Due mainly to spills and leaks,

[1] Also named in the complaint as defendants in the case are a number of Chevron's affiliates and/or subsidiaries. For ease of reference, the Court will refer collectively to all of the defendants as "Chevron."

Gulf's operation of the refinery resulted in the release of millions of gallons of gasoline and diesel fuel into the groundwater. Contaminated groundwater has subsequently migrated from the refinery site under Hooven. These Plaintiffs, however, do not claim injuries resulting from groundwater contamination. Rather, Plaintiffs claim injuries allegedly caused by air emissions from the refinery and, in particular, the benzene contained in those emissions.

Benzene is a solvent commonly found in many household products, such a paint and glue. Benzene is also ubiquitous in the ambient air and is a component or constituent of vehicle exhaust and cigarette smoke. In the petroleum industry, benzene is a component of gasoline. See generally Industrial Union Dept., AFL-CIO v. American Petroleum Inst., 448 U.S. 607, 615-16 (1980); United States Environmental Protection Agency, Benzene TEACH Chemical Summary, Toxicity and Exposure Assessments for Children (2009) (Doc. No. 415-2). While Chevron disputes the accuracy of Plaintiffs' estimate, it may be assumed for purposes of the present motions that the benzene content of the gasoline refined at the Hooven facility was 2.1%. Doc. No. 198-2, at 13. Benzene is a known carcinogen in sufficient doses. The issue in this case is whether Plaintiffs' benzene exposure, due to emissions from the refinery, was sufficient to cause their illnesses.

In estimating their benzene exposure, Plaintiffs employed a three-step procedure. First, expert Dr. Nicholas Cheremisinoff calculated a gross amount of benzene released from the refinery through emissions.[2] Then, using Dr. Cheremisinoff's calculations, Dr. Paul Rosenfeld, Plaintiffs' second expert, used an air flow model to calculate the cumulative dose of benzene to which each Plaintiff was exposed, a number expressed in micrograms ("ug"). Dr. Garabrant, Chevron's expert, converted Dr. Rosenfeld's microgram figures into the more conventional expression of parts per million per years ("ppm-years").[3] Finally, using Dr. Rosenfeld's dose estimates, Dr. Dahlgren has submitted opinions that each Plaintiff's dose of benzene was sufficient to cause her illness.

A. Carol Lipscomb and Jean Runck

Carol Lipscomb was born in 1939. She lived in Hooven from 1939 to 1956; in Harrison, Ohio from 1956 to 1957 and 1990 to 2000; in Cleves from 1957 to 1958 and from 1959 to 1965; in

[2] The Court notes that Chevron also disputes the reliability of Dr. Cheremisinoff's calculations. For purposes of the present motions, however, the Court assumes that Dr. Cheremisinoff's opinion on gross benzene emissions from the refinery would be admissible at trial.

[3] Expressing dose in ppm-years is easily understood. If a person was exposed to 1 ppm of a compound every year for 20 years, his or her total exposure to the compound would be 20 ppm-years.

Cincinnati from 1958 to 1959, 1966 to 1990, and from 2000 to the present. Doc. No. 375-8, at 2.

Jean Runck was born in Indiana in 1932. She lived in Hooven from the early 1930's up to 1936 and then again from 1937 to 1938. She lived in Addyston, Ohio from 1940 to 1943, North Bend, Ohio from 1944 to 1951 and 1966 to 1973, and Harrison, Ohio from 1961 to 1966. Otherwise, Ms. Runck has lived in Cleves and, in fact she has lived there since 1982. Doc. No. 375-8, at 9.

In September 2001, Ms. Lipscomb was diagnosed with monoclonal gammopathy of unknown significance ("MGUS"). Doc. No. 203, Ex. 6. Ms. Runck was diagnosed with MGUS in 2003. Doc. No. 203, Ex. 7. A gammopathy is "[a]ny disease in which serum immunoglobulins are increased[.]" TABER'S CYCLOPEDIC MEDICAL DICTIONARY (19th ed. 2001), at 810. MGUS is a disorder "marked by excessive levels of paraproteins in the blood." Id. MGUS is fairly common in elderly people. Doc. No. 252, at 31. MGUS is itself a benign condition although about 20% of the individuals with MGUS later develop multiple myeloma. Doc. No. 252-1, at 6. Multiple myeloma is a hematopoietic and malignant disease of the bone marrow "characterized by the infiltration of bone and bone marrow by neoplastic plasma cells." TABER'S CYCLOPEDIC MEDICAL DICTIONARY (19th ed. 2001), at 1345. MGUS requires monitoring rather than treatment. Doc. No. 252 at 48.

B. Mary Etta Brown Greener

Mary Etta Brown Greener was born in 1952. She lived in Cleves from 1952 to 1953 and in Hooven from 1953 to 1971 and from 1975 to 1976. Otherwise, except for a brief period when she lived in Addyston, Ms. Greener has lived and continues to live in Cincinnati. Doc. No. 375-8, at 5.

Ms. Greener was diagnosed and treated for Hodgkin's disease in 1972. Hodgkin's disease is a "form of malignant lymphoma characterized by painless, progressive enlargement of the lymph nodes, spleen, and general lymphoid tissue[.]" DORLAND'S ILLUSTRATED MEDICAL DICTIONARY (31st ed. 2007), at 542. Ms. Greener's treatment for Hodgkin's disease involved radiation therapy but not chemotherapy. Doc. No. 203, Ex. 9. Ms. Greener developed left breast cancer in 1999 and right breast cancer in 2000 and underwent radical mastectomies on both occasions. Plaintiffs theorize that emissions from the refinery caused Ms. Greener's Hodgkin's disease and that, in turn, the radiation therapy for Hodgkin's disease caused her breast cancers.

C. Michelle Schrader

Michelle Schrader was born in 1962. She has never lived in Hooven. Ms. Schrader lived in Cleves from 1962 to 1965, 1968, 1981, and 1991 to the present. She lived in North Bend from 1965 to 1966 and again from 1970 to 1980, in Sayler Park, Ohio in 1969, in Cheviot, Ohio in 1980, and in North College Hill, Ohio from 1982 to 1991.

Ms. Schrader was diagnosed with acute myelogenous leukemia ("AML") in 2000. Doc. No. 221-26. AML is "[a] group of hematological malignancies in which neoplastic cells develop from myeloid, monocytic, erythrocytic, or megakaryocytic precursors." TABER'S CYCLOPEDIC MEDICAL DICTIONARY (19th ed. 2001), at 1170. Ms. Schrader was treated with chemotherapy and her AML was reported in remission in 2004. Doc. No. 221-26, at 14.

II. Procedural Matters

Plaintiffs filed suit against Chevron in 2005 and assert tort claims under Ohio law which contend that their illnesses were caused by emissions from the Hooven refinery. On August 16, 2007, Magistrate Judge Hogan entered an order (Doc. No. 120) which established a trial calendar for resolving the claims of the instant bellwether Plaintiffs. In pertinent part, this order required Plaintiffs to provide their experts' reports by March 11, 2008. This deadline was later extended to May 30, 2008. Doc. No. 139.

Plaintiffs identified Dr. James Dahlgren, a medical doctor and professor of medicine at the University of California Los Angeles, as their medical causation expert. Dr. Dahlgren submitted expert reports on each of the bellwether Plaintiffs on or around May 14, 2008. Each of the reports follows the same format. There is a biography of each Plaintiff which summarizes their residential histories and provides some details concerning

their individual experiences living in and around Hooven. Included in the biography is a written summary of each Plaintiff's medical history and current or ongoing medical problems. Then there is a medical questionnaire reporting whether the Plaintiff had ever experienced any of a wide variety of symptoms. Dr. Dahlgren then reported the details of his own physical examination of the Plaintiff. Next, Dr. Dahlgren provided a very detailed review of each Plaintiff's medical records and laboratory tests, if any. Each report concludes with a brief section entitled "Discussion." The discussion in Ms. Schrader's report is representative:

> Michelle Schrader was exposed to benzene in the air and probably in the soil gas arising from the Chevron Refinery in Hooven. She was in utero and an infant in the neighborhood. She spent most of her young life in the area directly around the refinery.
>
> The patient's exposure from the refinery has resulted in the classic disease caused by benzene, namely acute myelogenous leukemia ("AML"). This patient's long-term exposure was essentially from birth up until 1982, and then from 1982 to 1991, she was probably out of the area. Her exposures, as documented by Dr. Rosenfeld indicate the presence of significant benzene and other cancer causing pollutants from the Chevron/Gulf Refinery. The dose as calculated with assumption of childhood adjustment by Dr. Rosenfeld are [sic] 3,218,823 ug of benzene, 5,079,747 PAHs cigarette equivalents (i.e. based on one microgram of PAHs per cigarette) of [sic] and 2,045,535,840 ug of total hydrocarbon exposures.
>
> Acute myelogenous leukemia is caused by benzene. Exposure to benzene is known to cause this particular type of cancer. The only other cause is radiation which is not a factor in this case.

> Studies of benzene exposure and leukemia do not show a threshold. That means to dater [sic] there is no study which has studied benzene and determined [a] level which does no [sic] increase the risk. The American Petroleum Institute published a report in 1948 which said there is no safe level of exposure. The only absolutely safe level is zero, they said. Subsequent studies have confirmed this finding. Ms. Schrader was exposed to benzene above the background level and that exposure occurred because of her residence in the vicinity near the Hooven refinery. There is no other reason she would have developed this devastating disease.
>
> She has required extensive medical treatment for her AML and will continue to require extensive medical care for the rest of her life. She is at increased risk for developing a second cancer because of her benzene exposure and her chemotherapy for the AML.
>
> The medical opinions expressed in this report are based on the information that was available to me at the time of preparation. I reserve the right to modify my opinion as additional information becomes available.

Doc. No. 221-27, at 4-5. Finally, attached to each report is essentially a bibliography or summary of medical studies on benzene exposure. Dr. Dahlgren did not, however, attempt to correlate or explain how his summary of the medical summaries supports his conclusion that benzene caused each Plaintiff's disease.

On June 26, 2008, Chevron moved to strike Dr. Dahlgren's reports concerning Lipscomb, Greener, and Runck on the grounds that they were not in compliance with Rule 26. Doc. No. 171. Plaintiffs then served Chevron with "supplemental" reports from Dr. Dahlgren dated July 7, 2008. Doc. No. 173. Dr.

Dahlgren's supplemental reports are essentially in the same format and Ms. Schrader's report is again representative:

> I examined Michelle Schrader on April 14, 2008. I prepared a report dated 05/14/08. In the report I state the following:
>
> *"Acute myelogenous leukemia is caused by benzene. Exposure to benzene is known to cause this particular type of cancer. The only other cause is radiation which is not a factor in this case.*
>
> *Studies of benzene exposure and leukemia do not show a threshold. That means to dater [sic] there is no study which has studied benzene and determined [a] level which does no [sic] increase the risk. The American Petroleum Institute published a report in 1948 which said there is no safe level of exposure. The only absolutely safe level is zero, they said. Subsequent studies have confirmed this finding. Ms. Schrader was exposed to benzene above the background level and that exposure occurred because of her residence in the vicinity near the Hooven refinery. There is no other reason she would have developed this devastating disease."*
>
> It is my opinion based on the information made available to me at the time of preparation that this woman's leukemia was caused by her exposure to benzene. I hold this opinion to a reasonable degree of medical probability. I reserve the right to modify my opinion as additional information becomes available.

Doc. No. 203, Ex. 11 (italics in original). On September 5, 2008, Magistrate Judge Hogan entered an order (Doc. No. 196) denying Chevron's motion to strike on the grounds that it was improper procedurally and that perceived deficiencies in the reports would be better addressed by means of a Daubert motion.

Chevron filed the instant motion for summary judgment and motion to preclude Dr. Dahlgren's opinions pursuant to

Daubert on September 12, 2008. On the merits of Plaintiffs' claims,[4] Chevron's motion for summary judgment is predicated on the asserted inadmissibility of Dr. Dahlgren's opinions. Chevron argues that Ohio law requires expert medical testimony to establish causation in a toxic tort case. Consequently, Chevron argues that if Dr. Dahlgren's testimony is excluded it is entitled to summary judgment on Plaintiffs' claims. In its Daubert motion, Chevron presents a number of arguments why Dr. Dahlgren's testimony is inadmissible. The principal argument raised, however, is that Dr. Dahlgren's opinions are unreliable because there is an insufficient scientific or medical basis to conclude that the doses of benzene to which Plaintiffs' were exposed were large enough to have caused their illnesses. Relatedly, Chevron contends that there is an insufficient scientific or medical basis to conclude that benzene even causes some of the illnesses, such as MGUS. The Court held an hearing on Chevron's Daubert motion on November 12, 2008 during which Dr. Dahlgren and Chevron's medical expert, Dr. David Garabrant, testified. Doc. Nos. 248 & 252.

Plaintiffs filed a memorandum in opposition to Chevron's motion for summary judgment and a memorandum in opposition to Chevron's Daubert motion on October 3, 2008. Doc.

[4] Chevron argues in the alternative that at a minimum it is entitled to summary judgment on Plaintiffs' claims for punitive damages.

Nos. 219 & 221. In their response to Chevron's Daubert motion, Plaintiff's submitted a new, 39-page affidavit from Dr. Dahlgren which expresses his opinions with considerably more clarity and detail that his original reports. Doc. Nos. 221-14, 221-15. Chevron then filed objections to Plaintiffs' evidence (Doc. No. 236), and in particular argued that Dr. Dahlgren's affidavit presented new opinions that were not disclosed before the deadline established by the Court.

While the parties were grappling with expert opinions, they were also litigating the adequacy of Chevron's privilege log before Magistrate Judge Hogan. On October 30, 2008, Judge Hogan entered an order (Doc. No. 241) requiring Chevron to produce 433 documents as not being privileged either because the privilege log did not put Plaintiffs on notice of the asserted privileged and/or the documents were non-privileged and discoverable. Plaintiffs filed objections to Judge Hogan's privilege log rulings. Doc. Nos. 242. Plaintiffs then moved to supplement their expert reports based on the documents produced pursuant to Judge Hogan's order on the grounds that they affected the calculations of their exposure to benzene. Doc. No. 249. Simultaneously, Plaintiffs moved the Court to review in camera a random sample of non-disclosed documents on the grounds that Chevron was abusing the attorney-client and work product

privileges to avoid having to produce damaging evidence. Doc. No. 250.

On December 19, 2008, the Court entered an order (Doc. No. 266) sustaining in part Plaintiffs' objections to Judge Hogan's order on Chevron's privilege log. The Court ordered production of about 50 additional documents but declined to conduct a further in camera review of a random sample of documents. The Court, however, did state that if the documents that were the subject of the order further suggested abuse of the privilege, Plaintiffs could renew their request for an in camera review.

The case came before the Court on December 18, 2008 for a final pretrial conference and on December 31, 2008 for a status conference. The main topics of discussion in these two conferences were whether Dr. Dahlgren would be permitted to testify at trial and whether an in camera review of Chevron's privilege log was warranted. Doc. Nos. 320 & 321. Ultimately, the Court determined that the entire privilege log should be reviewed and that Plaintiffs should be permitted to supplement their expert reports, if needed, based on documents produced pursuant to the review. The Court appointed a special master to perform the in camera review. The Special Master completed his review and provided a report on June 8, 2009. Doc. No. 357. As a result of the Special Master's review, approximately 270

additional documents were produced to Plaintiffs. Plaintiffs then filed supplemental expert reports and both parties filed supplemental memoranda concerning Dr. Dahlgren's opinions on July 20, 2009. Doc. No. 375 & 376.

Accordingly, the parties have had a full and fair opportunity to litigate the admissibility of Dr. Dahlgren's opinions.

III. Medical Causation

In a toxic tort case, the plaintiff must present evidence of both "general causation" and "specific causation." Terry v. Caputo, 875 N.E.2d 72, 76 (Ohio 2007). General causation establishes whether the substance or chemical at issue is capable of causing a particular injury or condition in the general population. Id. If the plaintiff establishes general causation, then she must establish specific causation. Specific causation establishes whether the substance or chemical in fact caused the plaintiff's medical condition. Id. at 77. In order to establish both general causation and specific causation, the plaintiff must present expert medical testimony. Id. at 74 syl. 2. Without expert medical testimony on both general causation and specific causation, a plaintiff's toxic tort claim will fail. Id. syl. 3.

IV. Standards for Admissibility of Expert Testimony

Pursuant to Federal Rule of Evidence 702[5] and Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 597 (1993), in order to be admissible, an expert's opinion must have a reliable foundation and be relevant to the case. The Sixth Circuit has explained that:

> Daubert attempts to strike a balance between a liberal admissibility standard for relevant evidence on the one hand and the need to exclude misleading "junk science" on the other. See Amorgianos v. Nat'l R.R. Passenger Corp., 303 F.3d 256, 267 (2d Cir. 2002). There is no "definitive checklist or test" for striking this balance, but the Supreme Court in Daubert set forth a number of factors that typically "bear on the inquiry." 509 U.S. at 593, 113 S. Ct. 2786. These include whether the theory or technique in question "can be (and has been) tested," whether it "has been subjected to peer review and publication," whether it has a "known or potential rate of error," and finally, whether the theory or technique enjoys general acceptance in the relevant scientific community. Id. at 594, 113 S.Ct. 2786. The Rule 702 inquiry is "a flexible one," and "[t]he focus . . . must be solely on principles and methodology, not on the conclusions they generate." Id. at 594-95, 113 S. Ct. 2786. An expert who presents testimony must "employ[ ] in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field." Kumho

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[5] Rule 702 states:

> If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise, if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

> Tire Co. v. Carmichael, 526 U.S. 137, 152, 119 S. Ct. 1167, 143 L.Ed.2d 238 (1999).

Best v. Lowe's Home Centers, Inc., 563 F.3d 171, 176-77 (6th Cir. 2009).

In this case, Dr. Dahlgren is offering causation opinions based on epidemiological studies. Epidemiology is the study of "the incidence, distribution, and etiology of disease in human populations." Federal Judicial Center, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (2nd ed. 2000), at 335. Epidemiology is usually the best evidence of general causation in toxic tort cases. Norris v. Baxter Healthcare Corp., 397 F.3d 878, 882 (10th Cir. 2005). The trial court, however, may exclude expert testimony based on epidemiological studies where the studies are insufficient, whether considered individually or collectively, to support the expert's causation opinion. General Elect. Co. v. Joiner, 522 U.S. 136, 146-47 (1997). The court may not exclude an expert's opinion because it disagrees with his conclusions. Nevertheless,

> conclusions and methodology are not entirely distinct from one another. Trained experts commonly extrapolate from existing data. But nothing in either Daubert or the Federal Rules of Evidence requires a district court to admit opinion evidence that is connected to existing data only by the ipse dixit of the expert. A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered.

Id. at 146.

V. Plaintiffs' Experts' Opinions

As indicated, Dr. Dahlgren's opinions were the end result of a three step process. First, Dr. Cheremisinoff calculated gross benzene emissions from the refinery. Second, Dr. Rosenfeld input Dr. Cheremisinoff's calculations into an air flow model endorsed by the U.S. Environmental Protection Agency called AERMOD to determine the lifetime exposure or dose of benzene to which each Plaintiff was exposed. Third, Dr. Dahlgren provided opinions that each Plaintiff's dose of benzene was sufficient to cause and in fact did cause her particular illness.[6]

A. Dr. Rosenfeld's Reports

In his initial report, Dr. Rosenfeld determined that Plaintiffs' most probable lifetime exposure to benzene was as follows:

Lipscomb	17,625,680 ug

[6] Plaintiffs' supplemental brief suggests that their personal injury claims are not limited to benzene exposure and that they also claim injury from polyaromatic hydrocarbon ("PAH") emissions from the refinery. Doc. No. 375, at 3 (stating that Hooven and Cleves were exposed to a variety of hydrocarbons, which included benzene, and that "at no time was this case ever about 'pure' benzene or 'gasoline' emissions."). That is something of a surprising statement given that the medical literature filed in this case nearly universally concerns benzene exposure in manner. Dr. Dahlgren does not provide any opinions in his supplemental report that exposure to PAH's was the cause of Plaintiffs' illnesses. Moreover, as Chevron points out in its principal Daubert motion, Dr. Dahlgren testified in his deposition that PAH's were not a cause of Plaintiffs' illnesses. Doc. No. 203, at 32. Thus, while this may not be a "pure" benzene case, benzene is surely the alleged causative agent in this case, not PAH's.

Brown Greener	24,407,503 ug
Runck	4,000,238 ug
Schrader	978,889 ug

Doc. No. 214-1, at 20. Additionally, because Plaintiffs were exposed to refinery emissions as children, and because cancer risks are higher from early-life exposures than similar exposures later in life, Dr. Rosenfeld calculated an adjusted exposure level based on the EPA's "Supplemental Guidance For Assessing Susceptibility From Early-Life Exposure To Carcinogens (2005)." According to Dr. Rosenfeld, Plaintiffs' adjusted lifetime benzene exposure was as follows:

Lipscomb	49,698,783 ug
Brown Greener	51,932,694 ug
Runck	12,777,006 ug
Schrader	3,218,823 ug

Id.

Because he expressed his calculations in micrograms, one of Chevron's experts, Dr. David Garabrant, converted Dr. Rosenfeld's unadjusted exposure calculations to ppm-years:[7]

Lipscomb	2.2 ppm-years
Brown Greener	3.1 ppm-years
Runck	.50 ppm-years
Schrader	.12 ppm-years

Doc. No. 203, Ex. 15, at 14. Additionally, using Dr. Garabrant's conversion method, though not reflected in the report,[8]

[7] The parties agree that Dr. Garabrant correctly performed this conversion. Doc. No. 252, at 12.

[8] The parties agreed in correspondence with the Court that it correctly employed Dr. Garabrant's methodology to convert

Plaintiff's childhood adjusted benzene exposure in ppm-years was as follows:

Lipscomb	6.2 ppm-years
Brown Greener	6.5 ppm-years
Runck	1.6 ppm-years
Schrader	.40 ppm-years

As stated, the production of additional documents from Chevron based on the Special Master's Report resulted in the creation of revised reports from Plaintiffs' experts. Dr. Rosenfeld's revised exposure calculations are as follows:

Lipscomb	10,948,785 ug
Greener	15,737,176 ug
Runck	2,684,005 ug
Schrader	380,625 ug

Adjusted for Childhood Exposure

Lipscomb	34,515,690 ug
Greener	33,789,880 ug
Runck	8,962,166 ug
Schrader	1,134,518 ug

Doc. No. 375-3, at 19-20. Converted to ppm-years, Plaintiffs' revised exposure levels are as follows:

Lipscomb	1.37 ppm-years
Greener	1.97 ppm-years
Runck	.335 ppm-years
Schrader	.048 ppm-years

Adjusted for Childhood Exposure

Lipscomb	4.37 ppm-years

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ug's to ppm-years . This correspondence is in the Court's files and will made available as needed if an appeal from this order is taken.

Greener	4.22 ppm-years
Runck	1.12 ppm-years
Schrader	.14 ppm-years

As can be seen, Plaintiffs' revised exposure levels are approximately 30% lower than the original calculations. Plaintiffs, however, shift their causation theory in their supplemental brief in support of admitting Dr. Dahlgren's opinions. Plaintiffs still contend that their cumulative exposure to benzene was sufficient to cause their illnesses. Plaintiffs now claim, however, based on Dr. Rosenfeld's amended report, that they were periodically exposed to short-term but intense levels of benzene which were more harmful than exposure to the same amount of benzene spread over a longer period of time.

B. Dr. Dahlgren's Original Opinions Are Not in Compliance with Rule 26

Regarding expert testimony, Rule 26(a)(2)(B) requires the disclosure of the expert's report and also mandates the requirements of the report:

> *Written Report.* Unless otherwise stipulated or ordered by the court, this disclosure must be accompanied by a written report--prepared and signed by the witness--if the witness is one retained or specially employed to provide expert testimony in the case or one whose duties as the party's employee regularly involve giving expert testimony. The report must contain:
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> (i) a complete statement of all opinions the witness will express and the basis and reasons for them;

> (ii) the data or other information considered by the witness in forming them;
>
> (iii) any exhibits that will be used to summarize or support them;
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> (iv) the witness's qualifications, including a list of all publications authored in the previous 10 years;
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> (v) a list of all other cases in which, during the previous four years, the witness testified as an expert at trial or by deposition; and
>
> (vi) a statement of the compensation to be paid for the study and testimony in the case.

Fed. R. Civ. P. 26(a)(2)(B). Rule 26(a)(2)(C) requires the parties to make expert witness disclosures "at the times and in the sequence that the court orders." Fed. R. Civ. P. 26(a)(2)(C). Failure to comply with Rule 26(a)'s disclosure requirements precludes a party from using such evidence at a trial or hearing, or on a motion, unless such failure was substantially justified or harmless. Fed. R. Civ. P. 37(c)(1); Rodgers v. Monumental Life Ins. Co., 289 F.3d 442, 450 (6th Cir. 2002); see also Roberts ex rel. Johnson v. Galen of Va., Inc., 325 F.3d 776, 782 (6th Cir. 2003) (stating that Rule 37(c)(1) requires "absolute compliance" with Rule 26(a)).

As indicated above, Dr. Dahlgren's initial reports on these Plaintiffs consists almost entirely of a recitation of their family and medical histories. Each report concludes with a brief "discussion" which apparently sets forth Dr. Dahlgren's

opinion that the Plaintiff's illness was caused by her exposure to benzene. Then, attached to each report is an "Appendix on benzene toxicity," which is essentially a bibliography and summary of a number of articles and medical studies on illnesses purportedly caused by benzene. With one exception, however, Dr. Dahlgren never explains in his report how the bibliography supports his conclusion that a Plaintiff's cumulative benzene exposure was sufficient to cause her disease. Therefore, Dr. Dahlgren's initial reports do not comply with Rule 26(a) because they fail to provide the "how and why" of his opinions. Salgado v. General Motors Corp., 150 F.3d 735, 741 n.6 (7th Cir. 1998) ("Expert reports must include 'how' and 'why' the expert reached a particular result, not merely the expert's conclusory opinions."); see also Smith v. State Farm Fire and Cas. Co., 164 F.R.D. 49, 54 (S.D.W.Va. 1995)(finding plaintiff's experts' reports not in compliance with Rule 26(a) where they "refer[red] to massive amounts of documents as the basis for the opinions which are expressed in vague terms, with few specific references.").

Dr. Dahlgren's causation opinion as to Michelle Schrader is more clearly stated but is no less perfunctory. Doc. No. 221-16. Dr. Dahlgren did cite a 1948 report issued by the American Petroleum Institute which states that there is no safe level of exposure to benzene, American Petroleum Institute,

Toxicological Review on Benzene (1948), at 2, but again he otherwise failed to explain how the remainder of the medical literature referenced in the appendix supports his opinion.[9]

On July 7, 2008, without leave of the Court and almost two months after the deadline established for filing expert reports, Plaintiff served supplemental reports from Dr. Dahlgren. Doc. No. 173. These supplemental reports, however, do not bolster the case for admitting his opinions. Each of the supplemental reports is one page in length. Doc. No. 203, Exs.

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[9] Dr. Dahlgren's reliance on the American Petroleum Institute's report essentially espouses the "one-hit" or "no threshold" theory of causation in which exposure to one molecule of a cancer-causing agent has some finite possibility of causing a genetic mutation leading to cancer. Federal Judicial Center, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (2000), at 407-08. While the one-hit theory has been accepted for purposes of establishing regulatory safety standards, it has not been accepted as a reliable theory for causation under Daubert standards. Allen v. Pennsylvania Eng'g Corp., 102 F.3d 194, 199 (5th Cir. 1996) ("Scientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiffs' burden in a toxic tort case."); McClain v. Metabolife Int'l, Inc., 401 F.3d 1233, 1240 (11th Cir. 2005) (holding that district court erred by not excluding plaintiff's expert's causation opinion because he neglected dose-response relationship); Henricksen v. ConocoPhillips Co., 605 F. Supp.2d 1142, 1162 (E.D. Wash. 2009) (excluding expert's opinion pursuant to Daubert where "he presume[d] that exposure to benzene in gasoline can cause AML in any dose."); National Bank of Commerce v. Associated Milk Producers, Inc., 22 F. Supp.2d 942, 961 (E.D.Ark. 1998), aff'd, 191 F.3d 858 (8th Cir. 1999); Sutera v. Perrier Group of Am., Inc., 986 F. Supp. 655, 667 (D. Mass. 1997). Moreover, since benzene is ubiquitous, causation under the one-hit theory could not be established because it would be just as likely that ambient benzene was the cause of Plaintiffs' illnesses.

10-13. The supplemental reports quote what purports to be the causation opinion from the original report. Then Dr. Dahlgren states that it his opinion that the Plaintiff's exposure to benzene caused her disease and that he holds this opinion to a reasonable degree of medical probability. Again, Dr. Dahlgren made no effort to connect the medical literature to his opinions.

It was not until October 3, 2008, five months after the deadline for filing expert reports, after Chevron took his deposition, and after Chevron moved to exclude his opinions, that Plaintiffs filed a 39 page affidavit from Dr. Dahlgren that set forth opinions which he attempted to support by specific references to studies on benzene. Doc. No. 221-14, 221-15. At this particular point in the case, this affidavit is arguably the only report from Dr. Dahlgren which complies with the requirements of Rule 26(a)(2) and yet Plaintiffs provided no justification for providing this report five months late. There is nothing in this affidavit that could not have been presented to Chevron by the established date to serve expert reports. As Chevron accurately argues in its reply brief (Doc. No. 237, at 3-6), Dr. Dahlgren's affidavit is not a true rebuttal report. Rather, it constitutes an improper attempt to correct the weaknesses and improprieties of his original reports that were previously identified by Chevron in its principal Daubert motion. Brainard v. American Skandia Life Assur. Corp., 432 F.3d 655, 664

(6th Cir. 2005); Pride v. BIC Corp, 218 F.3d 566, 579 (6th Cir. 2000) (affirming district court's denial of series of motions to modify expert witness scheduling order and provide rebuttal reports as a "transparent attempt to reopen the Daubert hearing now that the weaknesses in [Pride's] expert testimony have been pointed out.") (brackets in original); Campos v. MTD Products, Inc., No. 2:07-CV-00029, 2009 WL 2252257, at *8 (M.D. Tenn. July 24, 2009 ("[A] party may not use the guise of "supplementation" to "ignore court deadlines, reopen discovery, find new facts, and generate new expert reports.")(some internal quotation marks omitted).

Moreover, the Court does not regard Plaintiffs' failure to provide detailed reports from Dr. Dahlgren by the deadline established by Magistrate Judge Hogan as being harmless. As the proponents of Dr. Dahlgren's testimony, Plaintiffs bear the burden of demonstrating that their failure to provide proper reports from Dr. Dahlgren in compliance with the Court's order was harmless. Johnson, 325 F.3d at 782. A violation of Rule 26(a)(2) will generally be harmless if it involves an honest mistake on the part of one party coupled with sufficient knowledge on the part of the other party. Vaughn v. City of Lebanon, 18 Fed. Appx. 252, 264 (6th Cir. 2001). Plaintiffs' failure to provide proper expert reports from Dr. Dahlgren by the expert discovery deadline cannot be dismissed as an honest

mistake. The requirements of Rule 26(a)(2) are very clear. Plaintiffs and Dr. Dahlgren were certainly capable of complying with these requirements, as his supplemental affidavit indicates. Nevertheless, on perhaps the most crucial issue of the case, Dr. Dahlgren's initial reports only vaguely indicate his causation opinions and fail to clearly set forth the basis for his opinions. This type of omission is almost per se prejudicial. Reed v. Binder, 165 F.R.D. 424, 430 (D.N.J. 1996)("Nothing causes greater prejudice than to have to guess how and why an adversarial expert reached his or her conclusion.").

Accordingly, the Court will not consider Dr. Dahlgren's affidavit in ruling on either Chevron's Daubert motion or its motion for summary judgment.

C. Dr. Dahlgren's Third Supplemental Report

With leave of the Court, Plaintiffs obtained new reports from their experts based on documents produced in conjunction with the Special Master's review of Chevron's privilege log. Dr. Rosenfeld determined that Plaintiffs' cumulative exposure to benzene was less than originally calculated although he also opined that Plaintiffs were exposed to peak doses of benzene in excess of certain regulatory levels on many occasions. Dr. Dahlgren's supplemental report still maintains that Plaintiffs' cumulative exposures to benzene were sufficient to cause their illnesses. Dr. Dahlgren also states

that Plaintiffs' peak exposures to benzene in excess of regulatory levels were worse than lower levels of exposure for a longer period of time. Dr. Dahlgren states that, according to Dr. Rosenfeld's report, there were times when Plaintiffs, and indeed all Hooven residents, should have been wearing air respirators when regulatory levels of benzene emissions were exceeded. Dr. Dahlgren cites nine papers, which the Court addresses below, in support of his supplemental report and opinions.

Initially, the Court notes that to the extent that Dr. Dahlgren relies on the fact that Plaintiffs' illnesses were caused because they were exposed to benzene in excess of regulatory levels, his opinions are not admissible. The mere fact that Plaintiffs were exposed to benzene emissions in excess of mandated limits is insufficient to establish causation. Nelson v. Tennessee Gas Pipeline Co., 243 F.3d 244, 252-53 (6th Cir. 2001); David L. Eaton, Scientific Judgment and Toxic Torts-A Primer in Toxicology for Judges and Lawyers, 12 J.L. & Pol'y 5, 39 (2003) ("[R]egulatory levels are of substantial value to public health agencies charged with ensuring the protection of the public health, but are of limited value in judging whether a particular exposure was a substantial contributing factor to a particular individual's disease or illness."). This is because regulatory agencies are charged with protecting public health and

thus reasonably employ a lower threshold of proof in promulgating their regulations than is used in tort cases. Allen, 102 F.3d at 198.

Moreover, Dr. Dahlgren did not use Dr. Rosenfeld's supplemental report in a reliable manner in opining that Plaintiffs' exposures in excess of regulatory levels were sufficient to cause their illnesses. Dr. Dahlgren only cites one specific portion of Dr. Rosenfeld's report in support of this aspect of his revised opinions. Dr. Dahlgren states that:

> In 1977, the benzene levels [n]ear the laboratory at the fence line exceeded the NIOSH [National Institute of Occupational Safety and Health] STEL [Short Term Exposure Level] 316 times and the PEL [Permissible Exposure Level] 296 times. The ATSDR [Agency for Toxic Substances and Disease Registry] and EPA short term level is 0.009 PPM. That means the plaintiffs were exposed hundreds of times to levels more than 111 times the level set for short term community exposure and 33 times that set for chronic exposure. The highest one hour levels reached over 12 mg/M^3 or 400 times the acute Maximum Recommended Level (MRL) of .03 mg/M^3.

Doc. No. 375-2, at 4-5. In reaching this conclusion, however, Dr. Dahlgren failed to note that, according to Dr. Rosenfeld's report, not only did none of the Plaintiffs live near the refinery fence line in 1977, none of the Plaintiffs even lived in Hooven in 1977. Doc. No. 375-8 (indicating that in 1977, Lipscomb lived in Cincinnati, Greener lived in Addyston, Runck lived in Cleves, and Schrader lived in North Bend). In fact, as indicated earlier, Plaintiff Schrader has never lived in Hooven. Thus, there is no factual support in the record for Dr.

Dahlgren's conclusion that Plaintiffs were actually exposed to these excessive levels of benzene in 1977 and that such exposure caused their illnesses. See In re Scrap Metal Antitrust Lit., 527 F.3d 517, 530 (6th Cir. 2008) (holding that to be admissible under Rule 702, the expert's opinion must find factual support in the record).

The Court now turns to the literature cited in Dr. Dahlgren's supplemental report.

A. The Bois and Paxman Study

In 1991, Bois and Paxman reported the results of an animal study in which they exposed rats to benzene based on the 8 hour PEL limit and 15 minute STEL limit established by OSHA. Frédéric Y. Bois & Dalton G. Paxman, An Analysis of Exposure Rate Effects for Benzene Using a Physiologically Based Pharmakinetic Model, 15 REG. TOXIC. & PHARM. 122 (1991). Their study determined that rats exposed to benzene for a single peak exposure of 32 ppm for 15 minutes on average had a 20% higher increase in certain metabolites than rats exposed to 1 ppm of benzene for 8 hours. This paper, however, does not support Dr. Dahlgren's supplemental opinions. It does not report a dose-response relationship between benzene and any hematopoietic illness. Indeed, the authors state that "[w]hether the amount of metabolites formed actually presents an increased risk of leukemia remains to be seen." Id. at 134. While the authors write that their study

suggests that a peak exposure to benzene for a short period is more harmful than a lower exposure for a longer period time, it is clear that the purpose of the study is to consider whether the OSHA STEL is sufficiently protective.[10] As just discussed, regulatory agencies use a much lower threshold of proof in setting safety standards and the authors themselves note that "[p]olicy . . . must often be made with incomplete information." Id. Therefore, this study does not support an opinion that Plaintiffs' short-term peak exposures to benzene probably caused their illnesses.

B. The Glass Study

In 2003, Glass, et al., reported the results of their study of a cohort of Australian petroleum workers who had occupational exposure to benzene. Deborah Glass, et al., Leukemia Risk Associated With Low-Level Benzene Exposure, 14 EPIDEMIOLOGY 569 (2003). Initially, it should be noted that this study clearly does not support Dr. Dahlgren's causation opinions as to Plaintiffs Lipscomb, Runck, and Greener. The authors state that "[n]o association was found between non-Hodgkins lymphoma or multiple myeloma and benzene exposure[.]" Id. at 569; see also id. at 572 ("No increase in risk for non-Hodgkins lymphoma/ multiple myeloma was found with increasing exposure to

[10] In fact, the authors concluded that the OSHA STEL was sufficiently protective of workers.

benzene."). Moreover, this study does not support Dr. Dahlgren's causation opinion as to Plaintiff Schrader. The study did not find a statistically significant increase in leukemia among the cohort until cumulative lifetime exposure of benzene was in the 2 - 4 ppm-years range. Id. at 573.[11] In this case, Schrader's cumulative benzene exposure was .048 ppm-years or .14 ppm years if the childhood exposure adjustment figure is accepted. Thus, Schrader's cumulative benzene exposure was anywhere from 14 to 42 times less than the lowest cumulative benzene exposure associated with leukemia as reported by Glass.[12] As indicated by the Court

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[11] A study "is considered 'statistically significant' only when the odds ratio is expressed with a 95% confidence interval (consistently) and when that interval does not include an odds ratio of 1.0 or below." Smith v. Wyeth-Ayerst Lab. Co., 278 F. Supp.2d 684, 691 n.8 (W.D.N.C. 2003). In the Glass study, the odds ratio at less than 1 ppm-years was 1.0. The odds ratio at 1-2 ppm years was 3.9, but had a 95% confidence interval of 0.9-17.1. Thus the results for an increase in leukemia at these levels were not statistically significant. See also Turpin v. Merrell Dow Pharm., Inc., 959 F.2d 1349, 1353 n.1 (6th Cir. 1992) (instructing that where the "confidence interval includes relative risk values both less than and exceeding 1.0, the null value, a researcher cannot state that the results are statistically significant.").

[12] Interestingly, at the next interval, 4-8 cumulative ppm-years, the increase in risk of leukemia was not statistically significant, with a confidence interval of 0.4 to 13.6. See id. Glass did not explain or even theorize why the risk of leukemia would be higher at 2-4 ppm-years than at 4-8 ppm-years. The risk became statistically significant again at 8-16 ppm-years of cumulative exposure. Arguably, then, Glass does not support a causation theory on leukemia until cumulative exposure levels reach 8 ppm-years, or about 57 times higher than Schrader's cumulative exposure to benzene.

in Joiner, "there is simply too great an analytical gap between the data and the opinion proffered." 522 U.S. at 146.[13]

C. Collins

Collins, et al., studied 4,417 workers who were occupationally exposed to benzene while working at a Monsanto chemical plant in Sauget, Illinois. J.J. Collins, et al., Lymphohaematopoeitic Cancer Mortality Among Workers Exposed to Benzene, 60 OCCUP. ENVIRON. MED. 676 (2003). The study considered whether peak exposures to benzene increased the risk of the cancer. At the outset, the Court observes that this study does not support Dr. Dahlgren's causation opinion as to Plaintiff Greener because the authors found that while deaths from non-Hodgkins lymphoma were greater than expected, they were not associated with either peak or cumulative benzene exposures and, indeed, probably not related to benzene at all. Id. at 678. The study itself found little increase in risk for any lymphohematopoietic cancers at all cumulative exposures except for multiple myeloma. The study did find slight increases in the

[13] Glass also reported a higher risk of leukemia for workers whose intensity of exposure to benzene was high. Id. at 573. As the Court appreciates this study, however, the intensity of exposure was actually the cohort's average annual exposure to benzene. See id. at 571 (indicating that exposure intensity was calculated by dividing cumulative exposure by duration of employment). It stands to reason that workers who had the highest cumulative exposure to benzene also had the highest intensity of exposure, as defined by Glass. Thus, Glass does not fit Plaintiff's causation theory based on burst or peak exposures to benzene.

standardized mortality ratio or SMR[14] for multiple myeloma at all levels of exposure to benzene, but the SMR did not exceed 2, and therefore was not likely to have been the cause of the disease, until cumulative exposures exceeded 6 ppm-years. See In re Joint Eastern & Southern Dist. Asbestos Lit., 52 F.3d 1124, 1128 (2nd Cir. 1995) (stating that "an SMR greater than 2.0 means that d [disease] was more likely than not caused by c [causal factor]."). Even at the >6 ppm-years level, however, the number of excess deaths was not statistically significant because the confidence interval included 1.0. See Collins, et al., at 676. Moreover, none of the Plaintiffs in this case had cumulative exposures of benzene above 5 ppm-years, even when adjusted for childhood exposure. Thus, this paper does not support Dr.

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[14] The district court in Massachusetts explained that:

> SMR, or standardized mortality ratio, in epidemiology is the ratio of observed deaths to expected deaths according to a specific health outcome in a population. The calculation used to determine the SMR is simple: number of observed deaths/number of expected deaths. The SMR may be quoted as either a ratio or a percentage. If the SMR is quoted as a ratio and is equal to 1.0, then this means the number of observed deaths equals that of expected cases. If higher than 1.0, then there is a higher number of deaths than would be expected under normal circumstances. Similarly, an SMR of 100 would mean that the risk in the study population is equal to that of the general population. For example, an SMR of 641 represents a relative risk of dying from a particular cancer that is 6.4 times greater than that of the general population.

Taylor v. Airco, Inc., 494 F. Supp.2d 21, 25 n.4 (D.Mass. 2007).

Dahlgren's causation opinion in so far as cumulative exposure to benzene is concerned.

The authors also reported more deaths than expected from all leukemias (SMR=2.7) and multiple myeloma (SMR=4.1) where peak exposures exceeded 100 ppm for 40 or more days. Id. Again, however, the results were not statistically significant because the confidence intervals all included 1.0. Id.

Accordingly, the Collins study is an insufficient basis to support Dr. Dahlgren's peak exposure theory of causation.

D. The Hunting Study

Hunting, et al., conducted a cohort study of garage mechanics employed by the District of Columbia to determine whether their exposure to benzene in gasoline increased the risk of leukemia and other hematological cancers. Katherine L. Hunting, et al., 52 OCCUP. & ENVIRON. MED. 673 (1995). The authors found an excess of lymphatic and hematopoietic cancers among the vehicle mechanics with an SMR of 3.63. Id. Like the Collins study, however, the results here were not statistically significant as indicated by the confidence interval (.075-10.63) and the authors themselves stated that the "study is limited by the small cohort size and small number of deaths." Id. at 677.

Moreover, there are several important distinguishing facts about the Hunting study which render it an unreliable basis for Dr. Dahlgren's opinions in this case. The authors were not

able to quantify the mechanics' level of exposure to benzene. Instead, they categorized exposure levels as "high," "medium," and "low." Therefore, the mechanics' exposure levels are imprecise. Id. at 675. Additionally, 297 out of the 338 cohort members were in the "high" exposure subgroup and contributed 90% of the person-years to the study. Thus, if anything, the Hunting study reflects the results of high exposures to benzene. In this case, however, Plaintiffs' exposure to benzene was much, much lower. Finally, Hunting reports that many members of the cohort regularly either washed their hands in gasoline or cleaned auto parts with gasoline; others siphoned gasoline by mouth. The authors noted that research had shown that "dermal contact with petrol or benzene can result in significant benzene absorption" which was "very relevant for our cohort members." Id. at 677. In this case, however, Plaintiffs' exposure to benzene was through air emissions from the refinery and did not include regular dermal contact with gasoline like the Hunting cohort.

Therefore, for all of these reasons, the Hunting study is an insufficient basis for Dr. Dahlgren's supplemental opinions.

E. The Infante Meta-Analysis

In 2006, Infante published a meta-analysis of cohort studies on the association between benzene and multiple myeloma. Peter F. Infante, Benzene Exposure and Multiple Myeloma: A

Detailed Meta-analysis of Benzene Cohort Studies, 1076 ANN. N.Y. ACAD. SCI. 90 (2006). A meta-analysis is "a method of pooling study results to arrive at a single figure to represent the totality of the studies reviewed." Federal Judicial Center, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (2000), at 380. Meta-analysis works well for pooling randomized experimental trials, but is more problematic when applied to non-randomized observational studies of the effect of toxic agents. Id. This is because methodological differences in observational studies are usually more pronounced than in randomized experimental trials. Id.

Initially, however, the Court observes that as the Infante meta-analysis addresses causation for multiple myeloma, it does not support Dr. Dahlgren's causation opinions as to Plaintiffs Greener and Schrader.

Infante's meta-analysis compiled seven individual benzene cohort studies. Of these seven studies, only one, Rinsky's 1987 study, found a statistically significant excess risk of multiple myeloma as a result of benzene exposure. However, according to Infante's meta-analysis of the seven studies, there were 22 observed deaths from multiple myeloma versus 13.37 expected. After an adjustment assuming a Poisson

distribution,[15] the meta-analysis produced a statistically significant SMR of 2.13 for multiple myeloma.

Assuming that the Infante meta-analysis does not suffer from the methodological problems highlighted by the REFERENCE MANUAL, a closer review of the individual studies shows that the subject cohorts generally had much higher exposures to benzene than Plaintiffs in this case. Therefore, the Infante paper does not support Dr. Dahlgren's opinion that Plaintiffs' MGUS was caused by their exposure to benzene.

DeCouflé, et al., studied a small cohort of chemical workers exposed to benzene and found a non-significant excess of multiple myeloma. Pierre DeCouflé, et al., Mortality among Chemical Workers Exposed to Benzene and other Agents, 30 ENV. RES. 16 (1983). This study did not report and in fact could not report on the workers' level of exposure to benzene due to the absence of historical records. It can reasonably be inferred, however, that the exposure levels in the study were substantially higher than those experienced by Plaintiffs in this case. The authors noted that the workers were exposed to multiple fugitive emissions as a result of process technology and quality control

[15] A Poisson distribution is "[a] probability distribution which arises when counting the number of occurrences of a rare event in a long series of trials. It is named after its discoverer, French mathematician and physicist Siméon Denis Poisson (1781-1840)." See http://www.thefreedictionary.com/Poisson+distribution (visited December 10, 2009).

procedures. The plant used approximately 5.8 million gallons of benzene each year. Moreover, the authors reported that, similar to the mechanics in the Hunting study, it was a common practice for the workers to wash their hands and tools with benzene and some workers actually siphoned benzene to take home. Thus, the authors suggested that "at least some workers received benzene exposures in excess of levels given off by the 'ordinary run' of the manufacturing processes." Id. at 17.

Rinsky, et al., found a statistically significant increase in multiple myeloma among workers exposed to benzene at three Ohio rubber plants. Rinsky, et al., Benzene and Leukemia: An Epidemiological Risk Assessment, 316 NEW ENG. J. MED. 1044 (1987). Rinsky reported, however, that the mean cumulative exposure for the case group was 254 ppm-years, or about 60 times higher than Plaintiffs' childhood adjusted exposure levels. In fact, the control group from Rinsky's study experienced a significantly higher average cumulative benzene exposure (50 ppm-years) than Plaintiffs. Id. at 1044-45.

Wong found a non-significant increase in multiple-myeloma among a chemical worker cohort exposed to benzene. O. Wong, An industry wide mortality study of chemical workers occupationally exposed to benzene II: Dose response analysis, 44 BR. J. IND. MED. 382 (1987). Wong constructed three exposure groups, with the lowest group having a cumulative exposure of

less than 15 ppm-years. Id. at 384. Wong also constructed two groups to assess peak exposures to benzene, continuous and intermittent. These two groups were further subdivided into low, medium, and high exposure levels; the continuous exposure group also had a "very high" peak exposure level. Two of the deaths from multiple myeloma were in the intermittent exposure group. In the intermittent group, the lowest peak exposure to benzene was less than 25 ppm, id. at 383, and only about 17% of this subgroup was exposed for less than one year. Id. at 384. On the other hand, about half of the intermittent group was exposed to benzene from 1 to 9 years. Id. Wong does not identify the exact exposure level of the two multiple myeloma deaths in the intermittent group, but the percentages indicate that it was unlikely that their exposures were less than one year. Therefore, it is fair to infer that the multiple myeloma deaths experienced significantly higher benzene exposures than Plaintiffs in this case.

Fu, et al., presented a cancer mortality study on one cohort of English shoe manufacturing workers and one cohort of Italian shoe manufacturing workers. Hua Fu, et al., Cancer mortality among shoe manufacturing workers: an analysis of two cohorts, 53 OCCUP. ENVIRON. MED. 394 (1996). Fu, however, was not per se a benzene study. Rather, Fu studied the association between various cancers and the workers' exposure to leather dust

and solvents. Fu reported an excess of multiple myeloma deaths among the Italian cohort exposed to solvents and no association between solvents and multiple myeloma among the English cohort. Id. at 396. Fu did note that the primary concern with solvents was benzene. Fu, however, did not have any data concerning the workers' exposure to solvents or the duration of their exposure except that the Italian cohort's exposure to benzene was likely to have been high. Id. at 397. Fu did not even know whether the glues used by the English cohort contained benzene. Id.

Yin, et al., found only one death from multiple myeloma and only one other case of multiple myeloma in a study of almost 75,000 workers in China who were exposed to benzene. Song-Nian Yin, et al., A Cohort Study of Cancer Among Benzene-Exposed Workers in China: Overall Results, 29 AM. J. IND. MED. 227 (1996). Yin reported no data at all on the cohort's level of exposure to benzene.

Finally, Bloemen, et al., reported a statistically insignificant three deaths from multiple myeloma among a cohort of approximately 2,300 workers exposed to benzene. L.J. Bloemen, et al., Lymphohaematopoeitic cancer risk among chemical workers exposed to benzene, 61 OCCUP. ENV. MED. 270 (2004). The average cumulative benzene exposure of the cohort was 39.7 ppm-years or about 10 times greater than Plaintiffs' cumulative exposure. Id.

at 273. Moreover, the data on "peak exposures were too sparse to be useful[.]" Id.

As this summary indicates, even assuming the validity of Infante's meta-analysis, the individuals in the subject studies generally had significantly higher exposures to benzene than Plaintiffs Runck and Lipscomb experienced in this case. Therefore, as is the case with the Glass study, it is too great an analytical leap for the Infante meta-analysis to provide a reliable basis for Dr. Dahlgren's revised causation opinions as to Runck and Lipscomb.

F. The Mehlman Papers

Dr. Dahlgren cites two papers by Myron Mehlman in support of his revised causation opinions. The first paper, however, is not an epidemiological study or a meta-analysis. Myron Mehlman, Benzene: a haematopoetic and multi-organ carcinogen at any level above zero, 9 EUR. J. ONCOL. 15 (2004). Instead, this paper, while summarizing the results of other benzene studies, is captioned as a editorial and advances the argument that there is no safe level of exposure to benzene above zero. As indicated, however, the no threshold or one-hit theory is not an accepted causation theory under Daubert. The second paper is similar to the first and also appears to be in the nature of an editorial calling for a reduction in the regulatory standard for exposure to benzene. Myron Mehlman, Benzene, a

multi-organ carcinogen, 13 EUR. J. ONCOL. 7 (2008). Since these are not epidemiological studies they are not reliable support for Dr. Dahlgren's causation opinions. E.g., McClain v. Metabolife Int'l, Inc., 401 F.3d 1233, 1240 (11th Cir. 2005)(holding that expert's opinions were unreliable where, in part, he relied on government health reports and consumer complaints to establish medical causation); Benkwith v. Matrixx Initiatives, Inc., 467 F. Supp.2d 1316, 1326-27 (M.D.Ala. 2006) (excluding causation opinion based on papers that were not epidemiological studies); Kilpatrick v. Breg, Inc., No. 08-10052-CIV., 2009 WL 2058384, at *7 (S.D. Fla. June 25, 2009) (excluding expert's opinion based on articles that were not epidemiological studies, including an editorial authored by expert).

G. Patel

In 2004, Patel, et al., published a paper on their study of cancer associated with exposure to gasoline vapors. Ami S. Patel, et al., Risk of Cancer as a Result of Community Exposure to Gasoline Vapors, 59 ARC. ENV. HEALTH 497 (2004). The Patel study presents a somewhat similar fact pattern to the overall circumstances of this case in that leaking underground storage tanks released 50,000 to 900,000 gallons of gasoline into the groundwater of the communities of Hazle Township and Hazleton, Pennsylvania. Indoor testing by the EPA in 366 homes affected by the plume found benzene levels ranging from <8.3 ug/m^3

(undetectable) to 140 ug/m^3. However, no cumulative exposure for the residents of these homes could be estimated because of the absence of historical data on air and groundwater concentrations and duration of exposure. Id. at 498. There was a statistically significant increase in leukemia in this population. There was not a statistically significant increase in non-Hodgkins lymphoma. Id. at 500-01. There were no multiple myeloma cases reported in this study. Patel estimated that the maximum benzene exposure was 2 ppm-years and that the average benzene exposure was .03 ppm-years. Id.

Patel, however, noted some significant limitations of this study. First, the study indicates that the population's exposure to benzene likely was higher than reflected by the test results because the samples were taken potentially years after peak exposure. Moreover, sampling was done after remediation efforts had taken place and likely did not reflect the actual cumulative benzene exposure. Thus, although the exposure number reported by Patel supports Dr. Dahlgren's opinion that Plaintiffs' cumulative exposure to benzene was sufficient to cause their illnesses, Patel's number clearly is not accurate. See id. at 501 (noting that "the benzene exposure was not well-characterized" at an individual level). In fact, it is reasonable to infer that the Hazleton population's exposure to benzene was much higher than reflected in the study. Second,

Patel did not have an appropriate control group for this study. Id. at 501. Studies without control groups are of limited reliability. McClain, 401 F.3d at 1253-54 (stating that case studies must be viewed with caution); Allison v. McGhan Medical Corp., 184 F.3d 1300, 1316 (11th Cir. 1999) (stating that case reports and case studies are "universally" regarded as insufficient to establish causation because they lack controls); Casey v. Ohio Med. Prods., 877 F.Supp. 1380, 1385 (N.D.Cal. 1995) (stating that "case reports are not reliable scientific evidence of causation, because they simply described reported phenomena without comparison to the rate at which the phenomena occur in the general population or in a defined control group; do not isolate and exclude potentially alternative causes; and do not investigate or explain the mechanism of causation"); Federal Judicial Center, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (2000), at 475 (stating that case reports without control groups must be carefully considered).

Therefore, the Patel study is an inadequate basis for Dr. Dahlgren's causation opinions.

G. The Reynolds Paper

In 2003, Reynolds, et al., published the results of their study of cancer incidence rates among children in California who were exposed to hazardous air pollutants. Peggy Reynolds, et al., Childhood Cancer Incidence Rates and Hazardous

Air Pollutants in California: An Exploratory Analysis, 111 ENV. HEALTH PERSP. 663 (2003). This paper does not reliably support Dr. Dahlgren's opinions. The authors studied cancer incidence rates among children younger than age 15. Id. In this case, however, although each of the Plaintiffs experienced emissions from the refinery as a child, none of them developed their illnesses until adulthood. The authors only broadly categorized cancers into leukemias and gliomas[16] although they did provide specific incidence rates for acute lymphoblastic leukemia ("ALL") and acute nonlymphocytic leukemia ("ANLL" also known as "AML"). The study reports a slightly increased relative risk for AML (1.46), which Plaintiff Schrader has developed, but that increase was not statistically significant. More importantly, this report studied cancer incidence rates among children and Plaintiff Schrader developed AML well into adulthood. Finally, as stated above, studies reflecting incidence rates of disease are of little worth in determining causation. Rider v. Sandoz Pharm. Corp., 295 F.3d 1194, 1199 (11th Cir. 2002). Thus, for several reasons this paper is not relevant to any of the causation issues at hand in this case.

[16] A glioma is "[a]ny neoplasm derived from one of the various types of cells that form the interstitial tissue of the brain, spinal cord, pineal gland, posterior pituitary gland, and retina." STEDMAN'S MEDICAL DICTIONARY (27th ed. 2000)

H. Summary

The Court recognizes that an expert's opinion does not have to be unequivocally supported by epidemiological studies in order to be admissible under Daubert. Knight v. Kirby Inland Marine, Inc., 482 F.3d 347, 354 (5th Cir. 2007). In this case, the opinions expressed in Dr. Dahlgren's revised report are based on a scattershot of studies and articles which superficially touch on each of the illnesses at issue. However, no depth of opinion is developed in any of the selected references as to any of Plaintiffs' illnesses. It appears that Dr. Dahlgren has not differentiated the cases in any way and has assumed that each reference supports his causation opinion on each illness. That clearly is not the case, as the Court's summation of the studies demonstrates.

More importantly, however, none of these studies supports an opinion that benzene can cause the illnesses from which Plaintiffs suffer at the extremely low doses or exposures experienced in this case. The subjects of the case studies cited by Dr. Dahlgren generally had much higher exposures to benzene than these Plaintiffs. Moreover, there does not seem to be any consistent level of support that benzene exposure significantly increases the risk of developing multiple myeloma or non-Hodgkins lymphoma. It is medically accepted that benzene exposure can cause AML; however, Dr. Dahlgren has not cited any paper finding

that a cumulative exposure of .14 ppm-years significantly increases the risk of developing AML. See, e.g., Henricksen v. ConocoPhillips, Co., 605 F. Supp.2d 1142, 1176 (E.D.Wa. 2009) (concluding that medical studies did not support an opinion that benzene can cause AML at a cumulative exposure of 8 ppm-years).

The Court, therefore, finds that Dr. Dahlgren's causation opinions are not reliable under the standards enunciated by Daubert and, consequently, inadmissible. Accordingly, Chevron's motion to exclude the opinions of Dr. Dahlgren is well-taken and is **GRANTED**.

VII. Summary Judgment

Since the Court has ruled that Dr. Dahlgren's opinions are not admissible, Plaintiffs are unable to establish a material issue of fact on the causation element of their personal injury claims. See Terry, supra, at 14. Accordingly, Chevron's motion for summary judgment is well-taken and is **GRANTED.** The claims of Plaintiffs Lipscomb, Runck, Schrader, and Greener are **DISMISSED WITH PREJUDICE.**

IT IS SO ORDERED

Date January 6, 2010

s/Sandra S. Beckwith
Sandra S. Beckwith
Senior United States District Judge